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APPEARANCES:
           IN THE UNITED STATES DISTRICT COURT
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             FOR THE NORTHERN DISTRICT OF
                                                                     2
                                                                         For Plaintiffs:
                                                                               LAW OFFICES OF LUNDY & DAVIS, LLP
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 3
             MISSISSIPPI, WESTERN DIVSION
                                                                               BY: KEITH PRUDHOMME, ESQ.
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                                   )
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                                                                               (337) 439-0707
             Plaintiffs.
                           ) No. 3:03C0-P-D
 6
                                                                               kprudhomme@lundydavis.com
                                                                     6
                                                                         For Defendants Beazer, Inc., and Koppers, Inc.:
 7
            VS.
                                                                     7
                                                                               WILDMAN, HARROLD, ALLEN & DIXON, LLP
     KOPPERS, INC., ET AL
                                                                               BY: ANTHONY G. HOPP, ESQ.
                                                                     8
                                                                               225 West Wacker Drive
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             Defendants.
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                                                                         For Defendant Illinois Central Railroad:
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               Santa Monica, California
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                                                                               (662) 455-1613
               Monday, August 1, 2005
                                                                               chris@uwbbr.com
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18
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     Reported by:
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23
    DIANA JANNIERE
                                                                    22
     CSR NO. 10034
                                                                    23
     JOB No. 912645
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EXHIBITS (Continued): 1 or two. DEFENDANTS PAGE 2 Q What you previously gave us is certainly 3 Cancer, Heart Disease, and Diabetes 203 3 comprehensive? in Workers Exposed to 2, 3, 7. 4 A I tried to be. I tried to include everything 8-Tetracholodibenso-p-dioxin 1052 5 **DNA Adducts in Normal Tissue Adjacent** 5 that is -- well, that is relevant to this case. And to Breast Cancer: A Review 1053 6 unfortunately, you never can quite do that. There is 6 7 always a question that arises that is not covered with 205 Characterization of a Major Aromatic 7 **DNA adduct Detected in Human Breast** 8 the articles, but I tried my best. 1056 Tissues 9 Q Let's start with deposition Exhibit 151. This 8 10 is the Stellman, S-T-E-L-L-M-A-N, paper, entitled Breast 206 Molecular Epidemiology in 11 Cancer Risk in Relation to Adipose Concentrations of 9 **Environmental Carcinogenesis** 1066 10 207 The Interaction Between Alcohol 12 organochlorine Pesticides and Polychlorinated Biphenyls Consumption and GSTM1 Genotype on 13 in Long Island, New York. 11 Polycyclic Aromatic Hydrocarbon-DNA 14 (Defendants' Exhibit 151 was marked Adduct Levels in Breast Tissue 1067 12 15 for identification by the court 13 16 reporter.) 14 17 BY MR. HOPP: 15 18 Q Do you have that paper? 16 17 19 Yes, I do. 18 20 Q Did you rely on the Stellman paper for purposes 19 21 of your opinions in this case? 20 22 A Well, I think -- I mean, let me just see if 22 23 this is a - yeah, I think that this is a paper that I 23 24 included because it found a correlation with PCBs and 24 25 breast cancer in the breast tissue. Page 867 Page 869 Santa Monica, California, Monday, August 1, 2005 1 1 It probably isn't as strong a piece of evidence 2 9:10 A.M. - 4:45 P.M. 2 as some of the other papers, but I am trying to notice 3 3 here which one of the PCBs that they found the JAMES DAHLGREN, M.D., 4 correlation with because the PCBs have a -- share some 5 having been duly sworn, testified as follows: 5 similar toxicities, as the other dioxin-like chemicals. 6 6 So insofar as this paper supports the notion **FURTHER EXAMINATION** 7 that dioxin-like chemicals cause, at least in some 8 BY MR. HOPP: 8 cases, an increased risk for breast cancer, it is 9 Q Good morning, Dr. Dahlgren. 9 relevant to our study. 10 Α 10 Q This paper, actually, appears to be part of the Q We are here for the fifth session of your 11 11 National Cancer Institute's Long Island Breast Cancer 12 deposition for the Grenada, Mississippi/Creosote 12 Study Project; is that right? 13 litigation. 13 A That's correct. 14 Last time we were working through some of the Is that an ongoing project? 14 15 studies that you referred to in your report and in a 15 I don't know. I actually think that they 16 supplemental bibliography that you gave me on May 9th of 16 probably stopped the study at this point. This was five 17 2005. years ago, this was published. I don't know for sure, 17 18 What I would like to do this morning is to 18 but I believe it was probably concluded. 19 continue with that. First, let me ask you: Have you 19 Q And were there other papers that came out of 20 done anymore work to -- do you have any additional 20 this Long Island study? 21 references that we didn't either see on the bibliography 21 Yes. First, it was an epidemiological study 22 last time or that are cited in your first report? 22 that showed an increase in breast cancer prevalence in 23 A Oh, gosh, I don't think so. I think that -- I 23 certain communities in Suffolk County where Stony Brook 24 don't remember now that I am sitting here that there are 24 is located, where one of the authors at least was from additional ones. As we may go through, I might have one 25 Stony Brook. Page 868 Page 870

1 And I think they were trying to figure out if 1 regarded as a cross-sectional 2 there were some factors in that community that would 2 surrogate for a continuum of 3 give rise to breast cancer. 3 exposures that may have been 4 There is also some studies that were done in 4 experienced earlier in life." 5 New York City where they found a link to organochlorine 5 Do you see that? 6 pesticides and breast cancer. And this was a follow-up 6 A Yes, sir. 7 7 on that. So they were looking at that set of compounds Q Do you agree with that statement? 8 as well. 8 A Yes, it is a problem. PCBs do have a long 9 Q In the Long Island study -- the study -- strike 9 duration in the body, but they do not necessarily. 10 that. 10 If you take a reading, say, in the year 1999, 11 in any of the other papers that have been you don't necessarily know what that persons level --11 12 studied -- strike that. Monday morning. relative level of any of these long-lasting compounds 12 13 In any of the papers that have been published 13 was the same 20 years ago or 30 years ago. regarding the Long Island study, did they identify any 14 14 What we know about the induction of cancer is other strong risk factors for breast cancer other than 15 15 that there is a phenomena called latency. So that the one or two congeners of PCBs that we see in the 16 16 usually the exposure that had occurred 10, 20, even 30 17 Stellman paper, if you remember? years prior, may be the most important. 17 18 A They looked at some other factors. There was a 18 Having said that, there is another mechanism by pesticide that they were concerned about in Suffolk... 19 19 which cancer risk can be increased and that was not 20 County in particular; but those studies, as I recall, addressed in this paper per se. 20 21 were not positive. They didn't find a linkage between 21 Q Which mechanism is that? 22 the pesticide and the breast cancer risk. 22 A That is the reduction of the immune system 23 So the cluster of diseases in that part of Long 23 making it more difficult for the body to fight off 24 Island were never fully explained. 24 cancer. And that has been put forward with dioxins and 25 Q In the Spellman -- I'm sorry. Stellman paper, 25 dioxin-like compounds as an important mechanism by which Page 871 Page 873 the only PCB congener in which they found -- for which there is an increased risk for cancer. 1 they found an increased risk of breast cancer was 183; 2 2 So that the exposure of the person may have 3 is that correct? 3 been in the 5 to 10 years before - 3 to 5 to 10 - 1 4 A That's correct. 4 mean, the rate at which cancers grow is relatively slow. 5 Q Look at the Abstract, they did not confirm a 5 Just in real broad round terms, the doubling 6 previously reported association between breast cancer time for lung cancer, which is the most well studied, is 6 7 and PCB congener 118; is that right? 7 probably in the range of four months and breast cancer 8 A Correct. 8 is probably similar. 9 And they also looked at the most abundant PCB Q 9 So that by the time it is large enough to be 10 congener and did not find an association there; is that diagnosed, it has probably been there for 2, 3, 4 years, 10 11 right? 11 depending on the growth of the tumor. 12 A That's correct. I believe that is correct. 12 And in this particular case, they had a number 13 Let me just double-check. 13 of cases that were under age 50. They had a total of 14 153, the most abundant di-ortho congener. It 14 34 percent of their population of cases were under 50 15 makes up nearly one-fourth of total PCBs in human 15 and 55 percent of the controls were under 50. The 16 indices, but didn't possess estrogenic properties. reason I am focusing on those is those might have been 16 17 Let's look at Page 1246. It is 1246. 17 more rapidly growing tumors. 18 A Yes. 18 Anyway, the mechanism by which dioxin-like 19 Q Next to Table 4, there is a statement. It 19 chemicals - like PCBs, and the dioxins we are looking 20 says, "A serious weakness of this study 20 at in this area -- induce cancer is not which causes 21 As with all case-control and 21 genetic damage, it is altering the mechanism, such as 22 prospective studies in which 22 chemical PAHs as to more active carcinogenic agents. 23 measurement of body burden is 23 No. 2 is reducing the immune system and 24 Made at a single time, is that 24 altering the ability of the body to kill cancer cells. 25 Such a measurement may at best be 25 And the third mechanism is by estrogenic or Page 872 Page 874

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other hormonal changes which influence, promote the growth or likewise co-inhibit the growth of tumors. So those three mechanisms are established for dioxin-like chemicals.

This particular paper really focused on the estrogenic properties which is - which is fine, but one of the problems which they did in this case, which ! think reduced its ability to detect the difference is they used patients with benign breast disease as controls. 250 patients with benign breast disease and 73 patients admitted for other surgical procedures.

They basically had used patients that had some already abnormality in their breast tissue as controls and I think that was not a good idea. It would have been much better to have patients totally free of breast disease. Free of any confounding problems associated with the mechanism that leads to benign breast disease that may well be the same mechanism that leads to the malignancy.

But, anyways, the paper is in the literature and I have included it in my group of papers because it does have a parallel. In spite of that weakness, it still shows the difference with this one PCB that we just talked about.

(Brief Recess.)

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they did and what the 25th and 75th percentiles were in 2 parts per trillion.

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Q Because these were PCBs and not dioxins specifically, this paper is analogous to your conclusions regarding Sherrie Barnes but not directly related; is that right?

A Correct. It just shows the tenancy of dioxin-like chemicals to induce breast cancer. This study by itself, of course, wouldn't be supportive of that, but when taken in context with everything else that we know about -- about dioxin and dioxin-like chemicals, I believe, it is supportive.

Q It wouldn't - this paper the Spellman strike that.

This paper, the Stellman paper would not be supportive because it is largely a negative study; right?

A Yes, except for that one association that you mentioned, there are a lot of other points that could be made about this paper.

Let me justimake one major point. And, I think, we made it in the last deposition. And I made the point that unless you look at genetic factors, your going to weaken your ability to see an effect.

In other words, we now know that there is what Page 877

BY MR. HOPP:

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Q Dr. Dahlgren, is benign breast disease itself considered to be a risk factor for breast cancer?

Α Yes.

Q Let's look at Page 1245 just the first sentence in the word -- I'm sorry, in the section Discussion, this is, again, the Stellman paper.

MR. PRUDHOMME: I'm sorry, which page? MR. HOPP: 1245, right underneath the heading Discussion, it says, "The present analysis

> For the Long Island population is consistent with numerous studies and other populations that have shown little association between OCC body burden and breast cancer risk." Do you agree with that statement?

A When you say "several," I guess, it is a question of what do you mean by that? But, yeah, there are some other studies.

Originally, the early studies showed a positive association and then there were a couple of more done subsequently, that did not find an association.

How did they measure body burden in this study? 23

They did a blood level for the chlorinated hydrocarbon pesticides. Shown on Table 2, which ones 25 Page 876

we call the gene/environment interaction.

This gene, slash, environment interaction?

Correct. So that what we now know if you really want to see an effect of a chemical, it is important to try to identify the various genetic predispositions that exist for the development of breast cancer.

And we talked about it, I think, in terms of PAHs in the last deposition. When they started looking at the specific genotypes or polymorphisms, as they called it, and then looked for environmental factors and then, let's say, the effect of cigarette smoke causing an increased risk of breast cancer was clearly present.

And I believe that, that will be true here with the organochlorines and the dioxins and the PCBs. And the second point is something we also -- that I want to emphasize is something that we talked about a minute ago.

They looked at these patients after the diagnosis was made. If you look at the animal studies for dioxin-like chemicals, it is most important to know what they were exposed to in early life. And that that sets in motion, changes that make an increased risk for breast cancer.

And that is based on animal studies, where if Page 878

you expose animals in utero or in the first infancy, the 1 context of the animal studies; is that right? first few months of life to the dioxins, there is an 2 A Yes, that is what I've said. increased risk of breast cancer. If you expose the 3 Q Let me show you what I have marked as Exhibit animals as adults to dioxin, there is not an increased 4 152. This is the Warner paper. It is entitled Serum 4 5 Dioxin Concentrations and Breast Cancer Risk in the 5 risk of breast cancer. 6 So timing of the exposure is important in 6 Seveso Women's Health Study. 7 7 breast cancer. So, in other words, this study here, (Defendants' Exhibit 152 was marked which has, you know, I think, some value because in 8 for identification by the court 8 spite of all of these problems, there was still an 9 9 reporter.) 10 association shown; but they ignored it for reasons that 10 BY MR. HOPP: 11 11 I am not sure. Q And forgive me for repeating, just for context. They said, well, you know, we did so many 12 12 Seveso is a town in Italy; correct? measurements, finding one measurement that stood out was 13 13 A Correct. not significant. I don't think that is really quite 14 Q And Ms. Warner indicated in July of 1976, there 14 accurate if you look at all of the evidence. 15 15 was an explosion of a trichlorophenol manufacturing 16 These PCBs, dioxin-like chemicals, have been 16 plant near the city; is that right? 17 shown in test systems to increase the risk of breast 17 A Yes. 18 cancers from the various mechanisms that I have 18 Q That resulted in the highest TCDD levels known 19 mentioned. 19 in human residential populations; right? 20 And therefore, I think their study in spite of 20 A No, I think that --21 their conclusion that it was not significant, I think 21 Q That is what she says? 22 viewed in the context of all of the evidence, it is 22 -- I think that is probably correct. There are 23 significant. 23 some other examples of higher levels in people but not 24 Q The genetic factors you talked about a moment 24 from a residential exposure. ago, you talked about various polymorphisms and the fact 25 25 Q We talked about Revage last time and there were Page 879 Page 881 that people with individual polymorphisms or specific 1 1 some even higher levels in that industrially polymorphisms may be more prone to contract a specific 2 exposed population? 3 disease if they are exposed to certain chemicals: is 3 A That's correct. 4 4 that right? Q Now, again, forgive me if we've covered some of 5 A Yes, the evidence is strong that that happens. this, but there has been a series of papers published on 6 Q And we talked about that a little bit before in 6 the Seveso population? 7 terms of PAH, and we have got several studies on that 7 A There have been, yes. subject with various PAHs and specific polymorphisms? 8 8 Q Many of them published by someone named 9 A Correct. 9 Bertazzi. 10 Q Are you aware of any literature that looks at 10 A Dr. Bertazzi has published several of them. 11 the issue of susceptibility in people with specific 11 Q And in the Seveso studies, they divided the 12 polymorphisms in the context of dioxin or PCB exposure? population into three zones; right? 12 13 A No, they mention it in this paper, but they did A Well, I am trying to remember -- three zones 13 not look at it. 14 14 which would be high, medium and low. 15 Q I mean, as far as you know, that is the work 15 Q Sure. Let's look at -16 that is yet to be done in the literature that is yet to 16 A A, B, and C; is that what they call them? 17 be delivered or deliverable? 17 Q This is page - take a look at Page 625 of the 18 A As far as I know, it has not been done. I have 18 Warner study, this is in the second column, the first 19 not come across a study like that, but everybody knows 19 paragraph that starts with the words, "On 10 July 1976," 20 we need to do it. 20 if you look down there a little bit, it says which was 21 Q And, again, just to follow up on what you said 21 divided into exposure Zones A, B and R; right? Those 22 a moment ago, you talked about prenatal exposure and 22 are the three exposure zones? 23 increased risk. 23 A Yes. Well, there is also the non-ABR. There

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actually four zones.

is -- that is what I was trying to remember. There is

C

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The work on prenatal exposure and in increased

risk in breast cancer, that really has been done in the

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1 You know, you can look at the 95 percent and Right. The papers uniformly referred to people 2 2 in these four zones; is that right? And this is 25 percent confidence interval and see the range. 3 3 nomenclature they adopted for the Seveso papers? Q Look right above the chart, it says, 4 A Right. They were going to follow these folks 4 "Although the total number of cases 5 when the money was made available for that purpose. 5 is small, the TCDD levels for women 6 You're right. They looked at what they thought 6 with breast cancer appear to be 7 7 was a surrogate of exposure of where they were. shifted to the right at the low end 8 8 Q By where they lived or where they lived at the of the cumulative distribution. At 9 9 time of the exposure? the high end of the distribution the A That's right. 10 shift is not apparent.* 10 And the idea, again, with Seveso was because it 11 What does that mean? 11 was a large amount of TCDD and it settled over the 12 A Well, if you look at - what he's got here is 12 community, it was ongoing exposure through daily living 13 the full cohort, you know, is the solid line. 14 and eating plants, vegetables, and things like that; is 14 Q Okay. 15 15 Α The cases are the little diamond shaped. that right? 16 Correct. 16 Q Got it. Α 17 Now --17 Q And he is looking at the fact that the lower 18 A it was a zone that was evacuated also -- maybe 18 end of the curve there is a light shift to the right and 19 that is a different issue. 19 if you were to connect those diamonds together, the line 20 Q Warner then looked at 15 women; is that right? 20 would not superimpose itself on the full cohort. 21 21 A Let's see, infants to 40 years of age and 76 Q Okay. Thenk you. 22 resided in the area, total number of characteristics of 22 So what it suggests is that at that end of the 23 breast cancer: 15 breast cancer cases were identified. 23 curve, the values tend to be a little higher. 24 The cohort was 981 women who matched that. 24 In other words, there is more women who have 25 Q 981 women matched the cohort description and 25 the level here, which it looks like about 50 to 100, Page 883 Page 885 among them, there were 15 breast cancer cases? 1 then women who have values above 100 because then it 2 2 A Yes. Actually, when they first started the looks like the diamonds match with the rest of the 3 study in March of 1996, they had 1271 eligible women: 3 population. 4 but they had a bunch that they couldn't locate or 4 The significance of that is not terribly 5 5 wouldn't participate. powerful because there is only 5 data points above 100 981, 80 percent eventually participated. So 6 there. Well, above 110. And I think that -- I would they had a 20 percent rate that they could not identify 7 not make too much out of it because of the small 8 numbers. or locate. 9 Q Look at Figure 1, this is on Page 627. Picking up on that point Page 628, right at the 10 10 All right. end, they talk about some of the issues with the study. 11 Q Figure 1, Page 627, this shows a graft 11 If you look really at the second to the last full 12 cumulative distribution of serum TCDD levels in the 12 sentence of the article, it says, "This 13 13 breast cancer cases; is that right? Result should be considered an early 14 A Yes, it starts off with the serum TCDD level. 14 finding because the number of cases 15 Right. 15 is small and the cohort is relatively 16 Q And it shows, basically, a curve; is that 16 young." 17 right? 17 Do you see that? 18 A Well, they start with the lowest and go to the 18 A Yes, I do. 19 highest. 19 Q Has there been additional papers published on 20 Q And, I guess, my_point on this is that it is 20 the Seveso women's health study since the Warner paper 21 not a linear diagram. It gives us sort of a backward 21 in 2002? 22 S-type curve; is that right? 22 A Not to my knowledge. A Well, this is just one of those cumulative 23 23 Again, let's look at the first page of Warner 24 distribution curves showing what the -- you know, 24 just to conclude. This is the second column under the 50 percent level would be. paragraph that starts with the date "10 July 76." It Page 884 Page 886

1 says, "Ten- and fifteen-year follow-up 1 do is quantify the other peaks. And I have been 2 Studies of the Seveso population 2 frustrated by the Seveso studies ever since the 3 found no increased risk for breast 3 beginning, 4 4 cancer incidence or mortality. And I remember one of my colleagues Dr. Copiro 5 However, after 20 years of follow-up, 5 was in Italy at tight he was a pathologist and he was 6 6 a statistically nonsignificant interested in these cases. 7 7 increased risk for breast cancer And he went to Dr. Patrosi and he asked him, 8 mortality emerged among women who 8 why he didn't do this. And Dr. Patrosi refused to 9 resided in zones A or B," and then 9 answer the question, which I found very strange. But 10 Does Warner present further evidence of a 10 anyway, a lot things about the Seveso studies - you 11 statistically significant association between breast 11 know, obviously, are important. 12 cancer and serum TCDD levels? 12 For example, we just talked about genetic 13 A Isn't that what this paper is about? 13 susceptibility and certainly, when this study was 14 Q I just want to make sure that is your 14 published in 2002, that whole issue of the gene 15 understanding. 15 environment interaction was well-known. And it would be 16 A Yeah, if you look at the results of Cox, 16 relatively easy for them to study that in these women. 17 Table 3 --17 Anyway, it is one of my frustrations. 18 Q Okav. 18 Q Let's look at deposition Exhibit 153. This may 19 A It shows a statistically significant increase 19 head us in the direction here we are talking about. 20 in the risk associated with TCDD levels at a risk --20 This is the Zhang, Z-H-A-N-G, paper? 21 relative risk of 2.1 with a statistical significant 21 (Defendants Exhibit 153 was marked 22 value of .05. And 4.5 is the confidence. 22 for identification by the court 23 Q What is the range of serum TCDD levels in the 23 reporter.) 24 women in the Seveso --24 THE WITNESS: Yes, 25 A 13 to 1960. 25 Page 887 Page 889 1 Q And that is parts per trillion? BY MR. HOPP: 2 A That's right. It's really, really high values. 2 Q And it is entitled Serum Polychlorinated 3 Q Certainly, higher than background exposures or Biphenyls, Cytochrome P-450 1A1 Polymorphisms, and Risk 4 say, the Dallas cohort that Dr. Schecter has published 4 of Breast Cancer in Connecticut Women. 5 on repeatedly; is that right? 5 Do you see that? 6 A That's right. 6 A Yes. 7 Let's look at --7 Q Did you rely on this paper for the purposes of 8 A They didn't do total TEQs. They just did 8 forming your opinions in this case? TCDDs. 9 9 A Yes. 10 Q Right. 10 Q What is the conclusion of this paper in a 11 A And I don't know why they did that. I mean, 11 nutshell? 12 obviously, TCDDs is an important contaminant here, but 12 A That there is an increased risk in those women 13 probably would help if they had done the full spectrum that have this subtype. 13 because that explosion just didn't expose them to TCDD. 14 14 Q And the subtype being, it is a particular type 15 It exposed them with the full range of chemicals in that 15 of polymorphism; is that right? 16 class. 16 A Yes, they had the 1A1, m1 and m4 genotypes, are 17 Q Do you know if any of the Seveso studies had 17 not associated with breast cancer risks, but the m2 was 18 looked at total TEQ? 18 associated with increased risk, making the point that I 19 A No, it looks like they always done TCDDs. It 19 made earlier. 20 is sort of maddening because by the time they do the 20 However, they really only looked at the one analysis for TCDDs, they can do the other congeners 21 21 sub -- the one variation, if you will. And there are 22 because they have done all of the cleanup steps and done 22 other important polymorphisms. And I predict that those all of the work. 23 23 will also be done as we go along. 24 Q They had the blood. 24 Q So this looked at one particular -- sorry. 25 A They had the blood samples and all they had to 25 Several polymorphisms and found an association Page 888 Page 890

looked at PCBs? with just one? 1 2 A That's correct. They looked at the CYP1A1. 2 A I'm sorry. I said ten. There were nine 3 That, I think, is the only polymorphism that they looked 3 congeners, but what they did was they totaled the nine 4 4 and just did the statistics against the totals. And if 5 5 you notice, that it is in the parts per billion range. And I am just saying that there are some others 6 that have been shown that seem to be important for 6 So they didn't have a very sensitive assay to 7 7 breast cancer risk as well, that they didn't look at, get into the parts per trillion range that you need to 8 8 but still very important paper along those lines that we do to get all of the various congeners of interest. Q They didn't compare individual congeners; they 9 9 have been talking about. 10 10 And what it was looking, though -- strike that. looked at totals? 11 What it was looking at was serum PCB levels, 11 A That's right. 12 that means the level of PCBs in the women's blood; is Q And these are all Caucasian women from 12 13 13 that right? Connecticut; correct? 14 A Yes. And unfortunately, they didn't do all of 14 A That's right. 15 15 the various congeners, as I recall. Let me just And is this a positive study? Did they find an double-check that. 16 association? 16 17 A Let's see. Q Which congeners did they do? 17 18 A I am looking forward to see what they did. 18 Q Let me direct you. I think this is Page 1181. 19 Yeah, they did the ones that are listed here. 19 Α Um-hmm. 20 I won't recount them, but they didn't do all of them. 20 The right-hand column, first full paragraph, 21 Q Where are they listed? 21 "In our stude, a significantly 🧦 22 A On Page 1178, in the 4th full paragraph in the 22 increased risk of breast cancer 23 23 right column, it says, "The following PCB Was found only for the CYP1A1 m2 24 genotype*? Congeners were measured," and they did 10-24 25 congeners. 25 A Right. I think, they -- lack of association Page 893 Page 891 Q I see. Just for the record then, they did 1 with serum PCB levels. And, again, they used benign congener 74, 118, 138, 153, 156, 170, 180, 183, and 187; 2 breast disease as controls. I think clearly is for the is that right? reasons I mentioned earlier is a mistake. 4 A That's correct. 4 Q I don't want to interrupt but just so we are 5 Q So they took blood samples from these women, 5 clear, a better way to do the study would be to use who were being treated for breast cancer and analyzed it 6 women who had not been diagnosed with any type of breast 7 for levels of those congeners; is that right? disease as a control population? A That's correct. 8 A Correct. You are really confounding the study Q And they also looked to see if they had this 9 rather significantly. 10 polymorphism in their gene; is that right? 10 Q And in the Zhang paper, it admits that it is a 11 A That's right. 11 drawback or a weakness in the study? 12 Q And they compared the levels of PCBs with the 12 A Yes, they do. But, again, though, I think people with the polymorphisms and then found whether or 13 13 probably most importantly, they - well, that is - that 14 not there was an increased risk? 14 may be probably the most important problem in terms of 15 A That's right. 15 not finding an association and why they wouldn't. 16 Now, in our case, that is the Grenada/creosote 16 Q All right. I'm sorry. Are you done? 17 litigation, have you looked for polymorphisms in the 17 A They go on to discuss the fact that they needed 18 blood of any of the people whose blood you sampled? 18 more PCB-detailed analysis to explain the link. 19 A No, I haven't had the access to that particular 19 Q And so the Zhang paper can best be described as 20 variable that I have found. 20 a step in the direction of evaluating the connection 21 Q So that data does not exist for the Grenada 21 between organochlorine exposure and various 22 cohort? 22 polymorphisms, but an incomplete step in that direction? 23 A No, I don't have that data. 23 A Yes, for those reasons. But may be some others 24 Q And, again, this is PCB as opposed to TCDD or 24 we can talk about, but those are the major ones. dioxin congeners; right? That is, the Zhang paper 25 Let's look at deposition Exhibit 154. This is Page 892 Page 894

1 know what they said. the Adami paper, A-D-A-M-I. It doesn't describe -- they simply say, 2 2 (Defendants' Exhibit 154 was marked 3 3 for identification by the court "At present, there are no biological 4 4 or ecological or analytic data reporter.) 5 5 THE WITNESS: Yes. available that can be utilized to 6 6 BY MR. HOPP: indicate that there would be a 7 7 Q Entitled Organochlorine compounds and dominance one way or the other where estrogen-related cancers in women. 8 8 that exposure level that can affect 9 9 This is a review article: is that correct? human cancer risks have been achieved 10 Yes, they are reviewing other papers. I don't 10 in any but the most unusual think there is original data here. I think this is sort 11 11 situations," but they don't really 12 12 of a meta-analysis type of paper. say what they mean. Q Let's talk about that. One of the papers that 13 13 So this is largely a negative study? 14 you did cite and we will get to, was, in fact, a 14 A Well, no, this is a review article. It is not 15 a study. 15 meta-analysis or described itself as a meta-analysis. 16 16 What is a meta-analysis? Q That's right. 17 17 A That is where they take the various studies. Α It, basically, cites the various papers. And I 18 let's say, there is 8, 10, 12 different studies of a 18 think that is probably its greatest value, is that it 19 subject, try to put the data all together. So all of 19 included a few papers that - what I find when I look at 20 the exposed or cases and all of the controls, thus, 20 these types of papers, is that they have a paper that I 21 increasing the power to detect a difference. 21 haven't seen before in their bibliography due to the 22 22 Often studies have only a small number of imperfect search engines that we have out there. 23 people. The power to detect and effect is less. So by 23 They know. But, obviously, their conclusion 24 doing a meta-analysis you increase your ability to do 24 was that their reading of the literature was if it 25 that. 25 didn't prove a link and they give their -- "Our summary Page 895 Page 897 1 Q And just as an example, there was a paper by 1 analysis," which they don't call a meta-analysis, but 2 Worberg that did that on TCDD, which was a famous 2 they call it a summary analysis of occupational exposure 3 meta-analysis; is that right? 3 of organochlorines. 4 A Yes, there are a number of papers like that. 4 The rate ratio of breast cancer for exposed was 5 Q So Adami looks at other papers that discuss 5 0.84, which is 84 percent, which would have suggested 6 organochlorine compounds and estrogen-related cancers; 6 that there is no excess. And for PCBs, it's 108 -- I'm 7 is that right? 7 sorry. It is 84 for PCB's and 108 for TCDDs. And then 8 A Right. 8 they say for DDE, it is 1.27. That is their review of 9 Q And in the Abstract, one of the things they say 9 the literature. is in humans in neither ecologic data, nor occupational 10 10 Q And those are really the marker compounds they 11 studies provide clear support for an association between 11 look at: TCDD, PCBs and DDE; is that right? organochlorine exposure and the occurrence of these 12 12 A Yes. And they don't necessarily -- this paper 13 cancers, and that is breast and endometrial cancers? was published in '95. So there has been quite a bit of 13 14 A That's right. That is what they say. 14 work since that time. But, you know, this paper was of 15 Q And then they conclude and this, again, is in 15 interest because it had a review of papers which is why 16 the Abstract. It states. "We conclude 16 I included it. 17 That available data do not indicate 17 Q So generally informative, but not supportive of 18 that organochlorines will affect the 18 causation in the case of Sherrie Barnes; is that right? 19 risk of these two cancers in any but 19 A It is not supportive. That is correct. 20 the most unusual situations." 20 Q Let's look at the next one. It says Ahlborg, 21 A That is what it says. 21 A-H-L-B-O-R-G, deposition Exhibit 155. 22 Q In what sort of situations did Adami and its 22 (Defendants' Exhibit 155 was marked 23 co-authors find an increased risk of breast cancer? 23 for identification by the court 24 A Okay. Let's see what did they find here that 24 reporter.) 25 was - I have to go back to the Discussion. I don't BY MR. HOPP: Page 896 Page 898

Q And the Ahlborg paper is entitled 1 Harvard University grant from the Chemical Manufacturers 2 2 Organochlorine Compounds in Relation to Breast Cancer. with the additional support of the Swedish Cancer 3 Endometrial Cancer, and Endometriosis: An Assessment of 3 Society. So that these two papers are really parallel. 4 the Biological and Epidemiological Evidence. 4 Q Does Ahlborg find an association between 5 This is another review paper; is that right? 5 organochlorine compounds and breast cancer? 6 6 Actually, it is a journal called Critical Review of A Well, they make the same conclusions they did 7 7 in their previous paper. The cancer causes and control Toxicology. 8 A By the way, we should go back to the Adami 8 paper and the critical review paper, I think practically 9 paper for one second. 9 cover the same ground. 10 10 Q Sure. I don't see that they have added much to the And point out that it was supported by the 11 11 Α discussion and they come to the same conclusion. That Chemical Manufacturers Association and further support 12 12 the rates -- let's see, do they have exactly the same from the Swedish Cancer Society. 13 rates? This is a little bit longer paper. 13 Q You think that indicates some lack of 14 14 Well, in fact, the Ahlborg paper looks not only 15 15 objectivity? at human epidemiology, but mouse studies and it looks at some of the animal studies. 16 A No. I think what -- it is interesting that 16 17 usually when industries sponsors a study they are 17 And I am looking to see if they do their negative. You know, it is just -- I can't say that 18 18 summary analysis, yeah. And then they do talk about in 19 there was necessarily bias, but why would someone write 19 vitro/in vivo animal studies in more detail. But they 20 a review paper. 20 also review some of the same papers -- the 21 21 Why wouldn't someone spend the money doing some epidemiological papers. 22 22 primary research, writing up a review paper when you Their conclusions go on for several pages. Any 23 have a nice big grant from the Chemical Manufacturer's 23 particular conclusion that you are interested in? I 24 Association, it seems to me, you want to collect some of 24 mean, the bottom line was they recommended, as they did your own data. We got how many authors on this paper? 25 in their other paper, that they looked at other end Page 899 Page 901 1 It's 1, 2, 3, 4, 5, 6, 7, 8 authors on the paper, which 1 points in breast cancer as being more likely to be -- to 2 means that somebody did a heck of a lot of work. 2 show a weak estrogen effect. 3 And So, therefore, it probably cost a lot of 3 Q Did you rely on the Ahlborg paper for the 4 money. And, you know, my feeling would be they should 4 purpose of your opinions in this case? 5 have invested some of that money on some of the issues 5 A As I said about the other review paper, review that we have discussed where we need more information. 6 papers are mainly useful for sort of pulling together 7 Q And one can bias a review paper by selecting 7 all of the references. And since they don't contain any 8. Ithe underlining papers to review in sort of a skewed 8 new data, the underlining papers are what you rely upon 9 9 manner; right? in reaching a conclusion. 10 A A. And B, what you do is criticize the 10 You don't usually rely upon the opinion of positive papers and you sort of give greater credence to 11 11 someone else who has written a review paper. It doesn't 12 the negative papers, which, you know, you can argue 12 mean you can't, I guess, but I think the real issue is these things back and forth, which, of course, people 13 13 to rely upon the data and see what the data shows. 14 do. I just -- I just think in passing, I thought it was 14 Q For an exercise like the one that you have gone 15 worth noting that. 15 through in this case, the primary research is the stuff 16 Q Okay. Let's move on to another paper, Exhibit you really going to want to look at in forming your 16 17 155. 17 opinions? 18 (Defendants' Exhibit 155 was marked 18 A That's right. 19 for identification by the court 19 Q And review papers are simply helpful to collect 20 20 the primary research? 21 THE WITNESS: That's correct. The same year, 21 Α That's right. 22 by the way. 22 Let's just look at the Abstract on Ahlborg. BY MR. HOPP: 23 23 This is towards the end of the Abstract. It says, 24 Q Right. 1995, it is the Ahlborg paper? 24 *The hypothesis that human exposure 25 It is basically the same work. It is the 25 to environmental levels of Page 900 Page 902

organochlorines would favor an estrogenic overactivity leading to an increase in estrogen-dependent formation of mammary or endometrial tumors is not supported by the existing in vitro, animal and epidemiological evidence." In the ten years since Ahlborg was published, are you aware of additional evidence that has come up that supports the notion that exposure to these organochlorines favor estrogenic overactivity leading to an increase in estrogen-dependent formation of mammary

A Yes, I think there are studies now that make it even clearer that there is a link, but you have to take into account the specific agents and you have to take into account time of exposure, and you have to take into account the genetic predisposition.

Q All of the things that you started talking about this morning?

cancers?

A Right. And in 1995, none of those variables were addressed in the various studies that were done, which is why you saw a weak associations as this author calls it or these authors.

Q Since '95, though, we have seen some papers
Page 903

last 40 years. While many other cancers have stayed the
same or decreased in frequency. The same is true for
prostate cancer which is increasing steadily and even
more marketably in breast cancer. Both of those are
felt to be hormonally-related cancers. And it is likely
that there are factors in our environment.

In fact, our causing this condition because when people move from low cancer risk countries to high cancer risk countries, they begin to approximate the host country or their destination country in terms of their risk.

And that has been shown, for example, with Japanese women. If they are of Japanese descent and they are raised in the United States, they have American breast cancer risks. If they are Japanese descent and raised in Japan, they have Japanese breast cancer risks showing that it is environmental. That it's a environmental factor.

Q Have American women who are raised in Japan, if there are enough of them, ever been studied?

A Never been guided. I don't think there are very many of them.

Q In a lot of the studies that I have read over the last couple of weeks and the ones that I have shown you, start with a sentence something like, there is this Page 905

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which indicate an increased risk of breast cancer with
particular congeners of dioxin or PCBs and other
congeners of PCBs or dioxins which have an
antiestrogenic effect; is that right?

A Yes. And that goes to this issue that we are talking about. You have to get to the specifics of the various congeners, A, but equally important, you have to also look at the time of the exposure, given the age in which they were exposed, which I think is very important. But then there is also the genetic issue as well. So you have to address all of those to explain what is going on.

This author and Adami both point out that when the evidence in animals shows clearly that these chemicals have an adverse effect on the body. And the teasing part, just the increased risk of, let's say, breast cancer, may not be the whole story. You need to look at all of the cancers and you need to look at the susceptibility factors — I mean, the stuff that we have been talking about.

Q And the fact is that this search for what environmental factors cause breast cancer is an ongoing effort; is that right?

A Yes. As you know, the rate of breast cancer has been increasing steadily in the United States in the Page 904

hypothesis that environmental factors lead to the increased risk of breast cancer.

And it appears to me at least that the hypothesis is still under study and we have not yet isolated what is causing this increased risk?

A I believe we've got some pretty good ideas and as we go through these papers today, it becomes clear that there are some very strong links that are shown both by animal and human studies. We may not have all of the elements.

In other words, if we don't do the right study, we are not going to get the right response. And I think the issue of environmental factors are causative of breast cancer is irrefutable. The question is what are the factors in our environment that are causing it.

As you can probably tell just by the number of studies that have been done, one of the leading factors is these organochlorines, including the dioxins and the PCBs; and the organochlorine pesticides.

And I think it is irrefutable that these are probably are playing some role in the process. Now, we are also going to be talking about PAHs, which is another ubiquitous environmental factor in our environment, which has increased in our society and as a result of our heavy use of internal combustion engines

Page 906

and heavy dependence upon various fuels to generate energy, that increased level of PAH exposure in industrial societies are quite common.

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There is additional factors that I think are important and have not been addressed by any of the studies so far. And a couple of animal studies have addressed it, but what if you have significant PAH and organochlorine exposure together, what is the breast cancer risk going to be in a population?

Certainly, the animal studies would suggest the phenomenon of the dioxins increasing the enzymes in the body to create more of the PAH toxic intermediates would strongly suggest that the two would interact to increase the risk of cancer; and in particular, the risk of breast cancer.

And we know also in the same 40 to 50 year period, that there has been a very large increase in lung cancer risk.

And obviously, there has been a linkage to cigarettes, but there is also probably other factors. And those factors have been identified as the urban factor; that is, that if you compare the cancer rates in Nebraska with the cancer rates in the industrialized portions of New Jersey or Texas, there is as much as 190-fold difference in risk.

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2 much of an exposure is enough to increase the risk and 3 3, what is the critical exposure window for humans? 4 Right, those are at least three of the issues. 5 Q Let's address one of those. This is deposition 6 Exhibit 156. It is the Anderson paper. It is entitled 7 Critical Exposure Window -- I'm sorry. Critical Windows 8 of Exposure for Children's Health: Cancer in Human 9 Epidemiological Studies and Neoplasms in Experimental

factors which lead to the increased risk; No. 2, how

Do you so see that?

12 (Defendants' Exhibit 156 was marked 13 for identification by the court

14 reporter.)

Animal Models.

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THE WITNESS: Yes.

16 BY MR. HOPP:

> Q It says, in the Abstract, "The evidence for exposures occurring during the preconceptional period that have an association with childhood or adulthood concers is equivocal is that right?

That is what it says.

24 Q Does this paper help bring any clarity to the 25 situation? That is, does it identify critical windows

Page 909

You can have certain census tracts where the risk of breast cancer is contestably small for lung cancer and you go into other areas and the rates are order of magnitudes higher. What is causing that? Well, that is called the urban factor, but it is clear it has something to do with environmental factors.

We talked, I think, in the first session of 8 Jyour deposition about childhood asthma just as an example, and the fact that someone who lives in a farm environment or a rural environment is by no means unexposed, that there are a lot of exposures in that environment as well?

A Yes. I am just talking about the cancer risks and the observed studies. I didn't say people on farms had no exposure.

Q No.

Α But the point is that there is a difference.

Q There is an observed difference between urban and nonurban environments?

A That's right. And the difference can be very large.

Q I want to go back to a statement. Will you agree that there are at least three issues that are under study with respect to this increased risk of breast cancer; that is, one, what are the environmental Page 908

1 of exposure for children's health?

2 A Yes.

Q And tell me what the conclusion is -

4 Α Well --

5 Q -- with respect to critical exposure.

They give a diethylstilbestrol example.

7 Q That is the DES; that was the morning sickness

8 drug?

9 A No, it was the threatened abortion drug, as I

recall. They would give women diethylstilbestrol if 10 11 there was some spotting or bleeding or contractions and

12 that sort of stuff. I don't believe they used that to

13 treat nausea, but that is my recollection of what DES

14 was given for.

Q Got you. It is no longer on the market, DES?

DES is no longer administered for that purpose.

17 I don't even think it is generally available as a

18 treatment for menopause.

19 Q Does the Anderson paper address organochlorines

20 or PAHs? 21 A Well, I don't recall if they used any of the

22 examples from that literature or not. They used PAH and 23 one of their examples was 712 dimethylbenz[a]anthracene 24 (DMBA).

25 And they talked about the sensitivity was Page 910

1 greatest at the end of gestation and the number of 1 opinion, that makes the evidence much less equivocal. 2 targets cells are highest. And let's see, what else? 2 In utero exposure is terribly important to to the ultimate outcome in those children who are so 3 And ENU is another chemical. 3 4 4 Q What is ENU? What classic chemical? exposed. What they showed, I think, was prematurity, 5 That is ethylnitrosourea. It is a non-PAH 5 low postgestational birth weights and some developmental problems associated with in utero exposure. 6 6 organochlorine, but it is a nitrosamine compound, 7 7 nitrosourea compound. Let's see. We have on page --So, I mean, the evidence is, I think, as they say in here in animals is quite clear. And there has 8 well, they are not numbered. 9 MR. PRUDHOMME: Some of them are cut off. 9 been additional evidence in humans that has 10 THE WITNESS: That's right. It looks like it 10 strengthened, I think, that point about the greater 11 susceptibility of the fetus because of its development, 11 might be page 11. Yeah. It says here -- it talks about 12 another PAH. 12 making it more vulnerable to the low level effects of 13 13 "Also in mice, the genetic ability to these environmental chemicals. 14 14 respond to inducers of cytochrome Q Has Perera or anybody else identified prenatal 15 P4501A1, which metabolizes PAHs, 15 in utero or perinatal exposure to PAHs as a risk for 16 16 determined numbers of lung and liver breast cancer later in life? 17 17 tumors induced by A 1 think it was shown for dioxin in animals 18 3-methylcholanthrene and DMBA [but, 18 only. I don't think there are human studies and I don't 19 interestingly, not benzo(a)pyrene 19 think that has been done in PAHs in animals, as I 20 20 (BP)], confirming that metabolic recall. 21 activation of carcinogens is a 21 Q Just as of right now or as of today or as of 22 limiting factor in transplacental 22 recently, just dioxins in animals; is that right? 23 23 carcinogenesis for at least some A That's right. 24 24 chemicals.* Q Does virus exposure increase the risk of 25 Q But the conclusion of the paper, let's look at 25 cancer? Page 911 Page 913 page, I guess, it is 13 of 25, where it says, "Gaps in 1 1 A In animals there is some evidence that there 2 knowledge." 2 are some viral-related cancers. Humans the evidence, I 3 A Yes. 3 believe, is really quite a bit less. 4 Q Where they basically say -- they do say. 4 Q Is that something that is under study 5 somewhere? I mean, are people looking at a connection "Although animal models have shown 5 6 that cancer risk can be increased 6 between viruses and an increased risk of breast cancer? 7 after exposure to certain potentially 7 A People have looked at that for 50 years. It is 8 hazardous agents --8 the most well-studied question, I think, that is out 9 preconceptionally, in utero, and 9 there. 10 perinatally -- in humans, much of the 10 Viral illness generally follows a communicable 11 evidence is equivocal" and it remains 11 disease pattern. There is a particular liver cancer in 12 so; is that right? 12 Africa that has been linked to a virus, but none of the 13 A No, that's not true. 13 -- and in the pattern of distribution of the disease is 14 Q Okay. 14 compatible with an infectious causation. 15 A I mean, Perera has several papers published in 15 But none of the studies that have been done in 16 the last five years, including one this year, that 16 other countries have ever been able to make a conclusive 17 clearly shows that cord blood PAH levels is associated 17 link with virus and cancers. with increased risk of various health problems. 18 18 And any cancers or just breast cancer? 19 Q That is Francesca Perera? 19 Basically, I think, it's - I mean, other than 20 A Correct. 20 your increased risk of getting cancer if you have got 21 Q And have you cited those papers in your 21 HIV, and that is different. That is an immune system 22 bibliography? 22 problem. 23 A I hope so, but if not, it was an oversight. 23 Sure, that is different. 24 The point being, this was published in 2000. And i 24 But the virus being the initiator or the 25 think there has been quite a few papers, at least in my promoter of human cancer, I just don't think that there Page 912 Page 914

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has been any evidence to support that notion at least 1 They used, I guess, in order to inhibit the growth of 2 not in the United States. 2 these cells --3 Q Okay. Let's look at Exhibit 157. This is the 3 Q They looked at a total concentration of 14 Arcaro, A-R-C-A-R-O, paper entitled Antiestrogenicity of 4 PAHs; right? Environmental Polycyclic Aromatic hydrocarbons in Human 5 They combined some together. Individual PAHs comprising the reconstituted mixtures. So they had 14 6 **Breast Cancer Cells; right?** 6 7 7 PAHs. (Defendants' Exhibit 157 was marked 8 for identification by the court 8 Q And actually, got it from the St. Lawrence 9 9 River in some sediment sample. reporter.) 10 10 THE WITNESS: Yes. A They got it from some sediment sample, yeah. I BY MR. HOPP: 11 think they purified it. Extracted it in some way. 11 12 Q - And this is an Arcaro study; is that right? 12 You know, "The most prominent 13 Molecular ions for the 16 US 13 A Correct. 14 Q So what Arcaro and co-authors were studying 14 **Environmental Protection Agency** 15 were cells in a petri dish; right? 15 (USEPA) Priority Pollutant PAHs" and then they 16 16 number the 16. And they then in Table 1, give you the A That's right. 17 Q. Let's turn to the Materials and Methods 17 concentration from the St. Lawrence River sediment of section, this is Page 116 under heading 2. 18 the various compounds and then compared it to a clean 19 A "Um-hmm. 19 place, I think, Kinderhook is supposed to be clean -Q It says, "Recombinant human ER" and then a 20 relatively clean. And here I think the levels are much 20 21 Greek symbol that I can't -lower. 22 A That's an alpha. 22 Q Kinderhook is another place in New York; right? 23 Alpha and beta? 23 Yes. And I think what they used was that as a 24 24 Beta. control. Kinderhook being relatively clean. Α "-- were obtained from Panvera -- " which looks 25 25 Q So they found that these compounds acted as an Page 915 Page 917 to be some sort of company in Madison, Wisconsin. 1 antiestrogen? 2 A That's right. 2 A I believe they inhibited the growth of these 3 What is that? What is recombinant human ER 3 cells. No, no, they did a whole cell competitive 4 4 alpha and beta? estrogen receptor binding assay. So, basically, they 5 A To be a little succinct here, I think they got 5 looked at a receptor binding assay using radioactive ER positive breast cancer cells. 6 hydrogen attached to the various -- let me see, is it 7 Q Oh. I see. 7 estrogen they used? 8 A And obtained them from this company that grows 8 Competitive binding assay was carried out with up various -- when you have cancer cell line, you can 9 the human estrogen receptor, alpha or beta, incubated keep it growing. Because if you take most normal cells, 10 with - E2, I think, is an estrogen compound, estradiol they won't keep growing in vitro, in a dish culture. It 11 or something is their control -- positive control. Yes, 12 will go through a period of time and then they will die. 12 17 beta estradiol. And so they exposed the cells to 13 Whereas cancer cells live on and forever, which estradiol or PAHs and then measured the binding of the 13 14 is one of the problems with cancer cells. They don't 14 two. 15 have -- they don't have a mechanism to stop them from 15 Q Okay. And their conclusion was? 16 reproducing. 16 The conclusion was that it was antiestrogenic 17 So they have got cells here with alpha and beta 17 and I think competitively bound with the estrogen estrogen receptors. This is a very succinct paper, 18 18 receptor. 19 which they don't necessarily tell you all of those --19 Q So how, if at all, does the Arcaro paper 20 Q They ordered cells from a company that cultures 20 support your opinions in this case? 21 them, as opposed to going out and harvesting cells from 21 A Well, it shows that PAHs binded the estrogen 22 a group of people; right? 22 receptor, which, of course, we have known from other 23 A Right. They bought the cancer cells. 23 assays. 24 Got it. And what did they conclude? 24 Your question is why did we include this in our 25 Their conclusion was that it was antiestrogen. 25 bibliography? Page 916 Page 918

1 .	0 1 4 11 - 1		1
1	Q Let's use that question.	1	know cancer is a multistep process.
2	A The findings indicate that "seven	2	Q And PAHs are generally considered to be
3	PAHs competitively displace estradiol	3	genotoxic; correct?
4	when added to cultures of human	4	A That's correct.
5	breast cancer cells may have	5	Q As opposed to organochlorines which are not
6	consequences for human health. It	6	genotoxic but induce some sort of enzyme production?
7	has been suggested that by the	7	A Yes. And the relevance here is that they
8	binding with the estrogen receptor	8	induced an enzyme that metabolizes the PAHs to their
9	carcinogens may accumulate in the	9	genotoxic or mutagenic toxic intermediates, where they
10	nucleus and result in increased	10	become apoxides and become more active in attaching to
11	mutagenicity. The PAHs examined in	11	the DNA.
12	this study are common environmental	12	Q So the binding to the receptor is one step in a
13	pollutants that are metabolized in	13	chain reaction?
14	the body and thus could potentially	14	A Well, that would be the implication of this
15	bind the estrogen receptor, resulting	15	paper, yes.
16	in both a suppression of estrogenic	16	Q And on Page 125, they talk about looking for
17	responses and a possible increase in	17	synergistic activity and state that they were unable to
18	mutations in specific target issues."	18	find any; is that right?
19	Q Okay.	19	A Well, let's see what they meant by - what
20	A Do you want me to explain what that means? Do		paragraph are you looking at?
21	you think you understand it?	21	Q Page 125, first full paragraph, "Recently
22	Q I think I understand it, but can you give it to	22	the issue of synergy among endocrine
23	me in layman's terms?	23	disrupting chemicals has received
24	A Well, these in vitro studies are mechanism	24	much attention? Although we did not
25	studies. And what this study showed is that the PAHs	25	look specifically for synergistic
	Page 919		Page 921
-			the second secon
١.	hind to the cetrogen recenter	١.	
1	bind to the estrogen receptor.	1	responses, the antiestrogenic
2	Q But we need to figure out based on additional	2	potencies of the reconstituted
2	Q But we need to figure out based on additional studies is what that means for human health; right?	2	potencies of the reconstituted mixtures in the focus assay were
2 3 4	Q But we need to figure out based on additional studies is what that means for human health; right? A In the context of everything else, we know that	2 3 4	potencies of the reconstituted mixtures in the focus assay were roughly an average of the
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C-H-A-L-O-U-P-K-A, paper titled Synergistic Activity of Q Have you ever worked on a lawsuit or evaluated 2 polynuclear Aromatic hydrocarbon mixtures as aryl 2 a case that involved an MGP? 3 hydrocarbon (Ah) receptor agonists. 3 A No, but I have read about manufactured gas 4 4 This is an in vitro study of human breast cells plants. 5 and an in vivo study of rats and mice; is that right? 5 And those were plants that were used back in 6 Α 6 the old days where we used to have gas lights that lit 7 7 the streets; right? And the results show that the PAH mixture 8 exhibited nonadditive synergistic activities; is that 8 Α Yes. 9 right? 9 Q The old gas lamps? 10 A Yeah, it studied the synergistic effects on 10 Yes. They made some natural -- well, some 11 PAHs on the Ah receptor. 11 volatile gas out of -- you know, out of crude oil or 12 12 This did find synergistic activity? coal. 13 13 Α Yes, it did. Rather marketably. Q In fact, they took, I think - my understanding is that they took coal and through some process, made 14 Q I'm sorry. I didn't hear you. 14 15 this gas, and then there was a residue, which was this Rather marketably. 15 16 And did they find them in all three media, if 16 tarry substance? 17 you will, studied both the human breast cells and then 17 A Tarry stuff, that's right. Similar to in the mice and the rats or did they find them in just 18 18 creosote. 19 19 one of those areas? Q Well, in fact, it was a residue from somehow 20 A Let's see, they used induction of 20 distilling the coal; right? 21 21 "Hepatic CYP1A1 mRNA levels A That's right. Same idea as the creosote. This 22 transformation of the rat cytosolic 22 distillate is pretty close to creosote. 23 Ah receptor to a complex which binds -23 Well, in fact, there are studies which examine 24 to a dioxin responsive element, and 24 25 the induction of EROD activity and 25 The composition of both, right. Which should Page 923 Page 925 1 antiestrogenicity in MCF-7 human 1 be in our list of papers here, I would hope. 2 breast cancer cells, and inhibition 2 And so what they did was they took this MGP PAH 3 of splenic plaque-forming cell 3 mixture and administered it to the cells in the rats and 4 response to both T cell-dependent and the mice and then they took some other PAH containing 5 independent antigens in B6C3F1 mice. 5 substance; is that right? 6 For EROD and CYP1a1 mRNA induction 6 Well, I think they used benzo[a]pyrene. Let me 7 and cytosolic transformation 7 see what did they use for the clean -- cleaner control. 8. : activities and immunosuppressive 8 The idea being they compared the activities of 9 9 effects, MGP-PAH mixture was the mixture to the control; is that right? 10 approximately 100 to 900 times more 10 A The control is benzo[a]pyrene which they 11 potent as an Ah receptor agonist than purchased from Sigma Chemical Company. 11 12 expected based on its benzo[a]pyrene 12 So it is kind of a pure PAH? A single PAH? 13 content.* 13 Α Single PAH. 14 And that means that one, two, three, four --14 And then they compared that activity of the 15 four of the effects were - were stronger in 15 multi-PAH mixture? synergistic. The synergistic activity was still there 16 16 Α That's right. 17 but lower for the antiestrogenic response in MCF cells. 17 And they found that the crude MGP PAH mixture 18 really 19-fold, as opposed to 100 to 900 times greater. 18 had more of an effect than the single PAH? 19 So all of the end points showed at a greater 19 Α Correct. 20 effect when they looked at the manufactured gas plant 20 And then did they compare it -- compare the 21 residues, as opposed to just benzo[a]pyrene content 21 activity of the MGP PAH mixture to the activity of 22 predicted. 22 TCDD? 23 Q Are you familiar with the manufactured gas 23 A Yes, they did. TCDD was their positive 24 plants? 24 control. 25 A Yes. 25 And, again, they found that the MGP PAH mixture Q Page 924 Page 926

had more effect than the TCDD, which is the positive 1 pesticides -- mostly insecticides of 2 2 control? organochlorine origin and phenoxy 3 3 A Where is this data? Here it is on Table 3, the acid herbicides -- and risk of 4 4 dose effect of the three. TCDD was -- it had -- TCDD cancer.* 5 had a greater effect than either the benzo[a]pyrene or 5 Do you see that? 6 6 the MGP. A Yes. 7 Q Did they study a synergistic effect between 7 Q And that is, basically, the conclusion of this TCDD and the MGP PAH mixture? 8 8 paper "some limited evidence"; is that right? 9 9 A I don't think so. It would have been a very A Yes. I mean, I think, those are the words he 10 interesting thing to do but they didn't. 10 used, yes. 11 Q And this is one of the things that you are 11 Q Does this paper and any of the sources that it 12 talking about in this case, which is the potential for 12 reviews, identify an increased risk of breast cancer 13 that synergy; is that right? 13 with exposure to pentachlorophenol or creosote? 14 A Yes. 14 A Let's see what he says about creosote. Some 15 Q Now, this study does not indicate any sort of 15 TCDD exposures -- the Adami paper, they quote. They 16 relative risk for breast cancer in humans; does it? It 16 also quote a paper - a study that is ongoing, has no is not that type of study. 17 17 results, where they mention breast cancer. I think 18 A No, that is not that kind of study. It is not 18 there is relatively few references to breast here. 19 addressing the issue directly. It is sort of a 19 The proof of the paper shows no excess risk in 20 mechanism paper, but I thought it was important because 20 farming where the exposures are varied. There was no 21 it talks about a mixture similar to creosote and how it 21 increased risk in another farmer study done in Sweden. 22 has an effect on the Ah receptor, which would add to the 22 Several farmer studies have mentioned breast and found 23 adverse effect caused by that mechanism. 23 no excess, not all of them studied breasts. 24 Q Right. So, generally, informative but not 24 Basically, this paper is not really - it 25 directly related to causation of breast cancer in 25 probably shouldn't even be included except that it is in Page 927 Page 929 1 humans; is that right? 1 the general category, talking about the TCDD and it 2 A Correct. mentions creosote in its -- it really doesn't have very 3 MR. HOPP: Let's take five minutes. much in the way of data on creosote. 4 (Brief recess.) 4 Q So do you think you should not have listed this 5 MR. HOPP: Let's mark this next Exhibit as 159. 5 as a paper supporting your opinions? 6 (Defendants' Exhibit 159 was marked 6 A Well, first of all, it's by and large not 7 for identification by the court 7 focused on breasts. We probably included it because of 8 reporter.) 8 some of the other cancers that would have applied to 9 BY MR. HOPP: some of the cases, not necessarily Sherrie Barnes. 10 Q Dr. Dahlgren, I am handing you Deposition 10 Q All right. Okay. Let's look at the next one 11 Exhibit 159, the Dich, D-I-C-H, study entitled 11 deposition Exhibit 160. This is the Firozi paper, 12 Pesticides and Cancer. And this is a review paper? 12 F-I-R-O-Z-I. And it is entitled Aromatic DNA Adducts 13 MR. PRUDHOMME: Do you have an extra copy? 13 and Polymorphisms of CYP1A1 and NAT2 and GSTM1 in Breast 14 MR. HOPP: I'm sorry. 14 Cancer, is that right? 15 THE WITNESS: Um-hmm. Yes, this would be a 15 (Defendants' Exhibit 160 was marked 16 review paper. 16 for identification by the court 17 BY MR. HOPP: 17 reporter.) 18 Q And they looked generally at various pesticides 18 THE WITNESS: Yes. 19 and cancer in animals and humans: correct? 19 BY MR. HOPP: 20 A Yes. 20 Q And this is a study of genetic polymorphisms 21 Q And the conclusion is -- looking at -- this is 21 and DNA adducts; right? 22 now Page 438, in the paragraph entitled Conclusion, it 22 A Yes. 23 says, "Epidemiologic studies provide 23 Q Tell me in layman's terms what the authors of 24 some, albeit limited, support of an 24 this paper - what question the authors of this paper 25 association between exposure to were trying to answer? Page 928 Page 930